Enhanced paracrine FGF10 expression promotes formation of multifocal prostate adenocarcinoma and an increase in epithelial androgen receptor.

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Public Summary:

Scientific Abstract:
Enhanced mesenchymal expression of FGF10 led to the formation of multifocal PIN or prostate cancer. Inhibition of epithelial FGFR1 signaling using DN FGFR1 led to reversal of the cancer phenotype. A subset of the FGF10-induced carcinoma was serially transplantable. Paracrine FGF10 led to an increase in epithelial androgen receptor and synergized with cell-autonomous activated AKT. Our observations indicate that stromal FGF10 expression may facilitate the multifocal histology observed in prostate adenocarcinoma and suggest the FGF10/FGFR1 axis as a potential therapeutic target in treating hormone-sensitive or refractory prostate cancer. We also show that transient exposure to a paracrine growth factor may be sufficient for the initiation of oncogenic transformation.